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Clinical management of a Veteran with chronic scan-negative cauda equina syndrome: a case report

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Objective: *To describe the clinical presentation, diagnostic considerations, and safety-first course of chiropractic care for an adult male with chronic scan-negative cauda equina syndrome.*

Clinical features: *A 41-year-old male U.S. Veteran sought chiropractic care for progressively intensifying chronic low back pain and bilateral lower extremity pain along with progressive bladder dysfunction, groin hypoesthesia, and sexual dysfunction. The history and examination findings raised concern for cauda equina syndrome, but a same-day MRI and neurosurgery consult failed to yield surgical indications.*

Prise en charge clinique d'un vétéran atteint d'un syndrome chronique de la queue de cheval sans anomalie à l'imagerie: rapport de cas

Objectif: *Décrire la présentation clinique, les considérations diagnostiques et le déroulement d'une prise en charge chiropratique axée avant tout sur la sécurité chez un homme adulte atteint d'un syndrome chronique de la queue de cheval sans anomalie à l'imagerie.*

Caractéristiques cliniques: *Un vétéran américain de 41 ans a consulté en chiropratique pour une lombalgie chronique d'intensité croissante et des douleurs bilatérales des membres inférieurs, accompagnées d'une dysfonction vésicale progressive, d'une hypoesthésie de l'aine et d'une dysfonction sexuelle. L'anamnèse et les constatations faites à l'examen ont soulevé la possibilité d'un syndrome de la queue de cheval, mais une IRM réalisée le jour même et une consultation*

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The involved patient provided consent for case publication.

Intervention and outcomes: *Chiropractic care involved 8 visits over 10 weeks. Care focused on neurological monitoring and patient education along with soft-tissue therapy, flexion-distraction, and spinal manipulation. The patient reported improvements in pain, function, and neurological features following this course of care, and no adverse events occurred.*

Summary: *This case emphasizes timely recognition of features associated with cauda equina syndrome and illustrates a cautious, multimodal approach to chiropractic co-management for an individual with scan-negative cauda equina syndrome.*

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KEY WORDS: pain, low back; cauda equina syndrome; chiropractic; manual therapies; Veterans health; veterans health services

en neurochirurgie n'ont révélé aucune indication chirurgicale.

Intervention et résultats: *La prise en charge chiropratique a comporté huit consultations réparties sur dix semaines. Elle était axée sur la surveillance neurologique et l'éducation du patient, et comprenait également un traitement des tissus mous, des techniques de flexion-distraction et des manipulations vertébrales. Au terme de cette prise en charge, le patient a signalé une amélioration de la douleur, de la capacité fonctionnelle et des manifestations neurologiques. Aucun événement indésirable n'est survenu.*

Résumé: *Ce cas souligne l'importance de reconnaître rapidement les manifestations associées au syndrome de la queue de cheval et illustre une approche multimodale prudente de la prise en charge chiropratique conjointe d'une personne atteinte d'un syndrome de la queue de cheval sans anomalie à l'imagerie.*

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MOTS CLÉS: lombalgie, syndrome de la queue de cheval, chiropratique, thérapies manuelles, santé des vétérans, services de santé aux vétérans

Introduction

Cauda equina syndrome (CES) is a serious neurologic condition characterized by dysfunction of the lumbosacral nerve roots within the spinal canal.[1,2] It typically manifests as a constellation of neuromuscular and urogenital features, including severe low back pain (LBP), lower extremity radicular pain or paresthesia, lower extremity weakness, gait disturbance, reduced perineal or genital sensation (i.e., saddle anesthesia), diminished reflexes, and bowel, bladder, or sexual dysfunction.[3] Although back pain, radicular leg pain, and lower extremity neurological deficits are frequently present, they are not required for diagnosis.[3]

While large disc herniations are the most common cause of CES (Figure 1), clinicians must also consider other compressive causes (e.g., neoplasms, hematomas) along with non-compressive etiologies (e.g., ischemia, infection).[1] The estimated incidence of CES in the general population ranges from 0.3 to 1 per 100,000 individuals.[4] Among patients with a history of lumbar disc surgery, CES occurs more frequently, accounting for approximately 2–6% of cases.[5] In primary care settings, CES is reported in approximately 0.08% of patients presenting with LBP, with a comparable prevalence suspected among adults seeking chiropractic care.[4,6] Magnetic resonance imaging (MRI) is the diagnostic gold standard for CES, as it provides definitive evidence of mechanical compression of the cauda equina nerves or other etiologic pathology.

The classification of CES typically depends on acuity: *acute* CES involves a rapid onset of symptoms, whereas *chronic* CES involves an insidious or gradually progressive clinical course.[7] Chronic CES is less understood and may arise from degenerative changes that progressively narrow the spinal canal, compromising nerves of the cauda equina.[8,9] While gradual compression may allow for

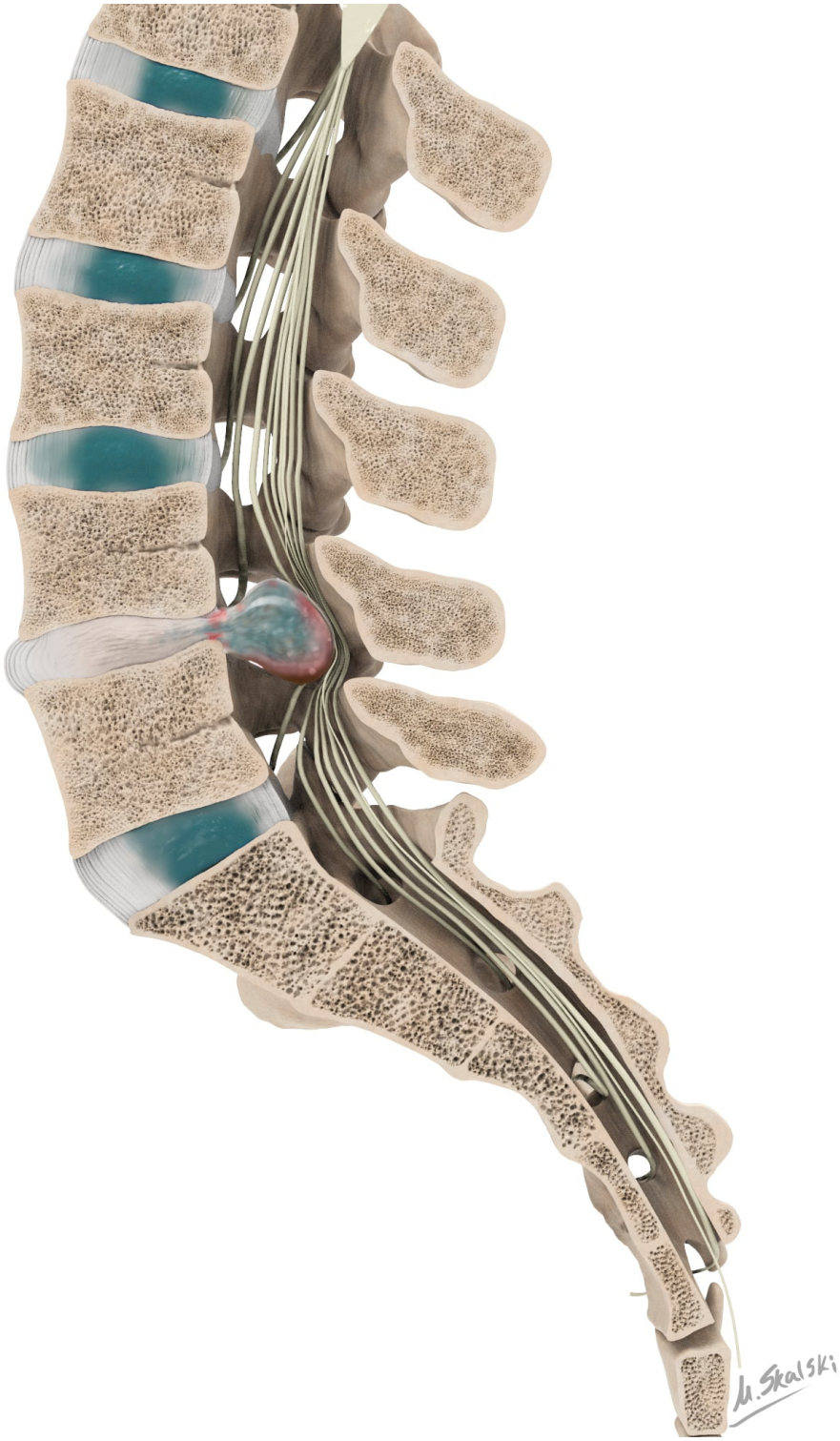


Figure 1.
Sagittal lumbar spine demonstrating a large L4 central disc herniation compressing the cauda equina.

early physiologic adaptation, persistent or progressive compression of the cauda equina eventually gives rise to the development of CES features.[10]

More recently, CES has been further classified based on imaging findings into *scan-positive* and *scan-negative* CES subtypes.[11] Scan-positive CES refers to cases in which radiologic evidence demonstrates compression of the cauda equina nerve roots consistent with clinical findings, whereas scan-negative CES describes patients with classic CES features in the absence of demonstrable cauda equina compression or causative etiology on MRI.[7,8] Scan-positive CES is typically considered a surgical emergency, with decompression within 24–48 hours associated with improved prognosis.[11,12] Since scan-negative CES lacks a structural target for neurosurgical decompression, surgery is not indicated. Consequently, scan-negative CES represents a distinct clinical entity that poses significant challenges regarding both diagnosis and management.

The purpose of this report is to highlight the red flag features that necessitated emergent referral and to illustrate a safety-first course of chiropractic care, characterized by continuous symptom monitoring and serial neurological assessments, with prioritization of red flag screening and graded application of patient-tolerated interventions for an adult male with chronic scan-negative CES.

Case presentation

A 41-year-old Caucasian male U.S. Veteran sought chiropractic care for LBP at a Veterans Affairs (VA) outpatient chiropractic clinic. His LBP was primarily located at his axial lumbosacral junction. It began insidiously about 15 years prior but had been gradually intensifying over the previous two years, to what he then described as an "excruciating" and "unbearable burning" sensation extending from his lumbar spine to his bilateral posterior thighs. His pain was worse with thoracolumbar extension and prolonged walking, while it was temporarily relieved with seated lumbar flexion-based stretching. He denied any history of invasive spine procedures or recent injuries, infections, or fevers. Baseline measures indicated high levels of distress and disability, with an 8/10 pain intensity and a Back Bournemouth Questionnaire score of 49/70.

Over the previous year, he described having experienced a gradual onset of intermittent periods of reduced sensation in his legs and groin (i.e., saddle anesthesia), and erectile dysfunction resulting from diminished sensation. Additionally, he explained how this coincided with the gradual onset of unpredictable episodes of urinary retention or urinary incontinence. He explained how his bladder dysfunction initially occurred about one to two times per week but described how this had gradually progressed to a nearly daily occurrence that had been ongoing for the 3-4 months prior to his chiropractic intake.

This Veteran also expressed growing concern for how his condition was negatively impacting his daily activities. He explained how his worsening symptoms, in combination with housing instability and his mental health conditions (i.e., PTSD, depression, and adjustment disorder), had resulted in a breakdown of his interpersonal relationships and social roles. He also noted how his pain and bladder dysfunction had caused substantial distress, which he felt had contributed to his desperation and prior attempts at suicide. These issues prompted his admission to an inpatient VA mental health rehabilitation program, which is where he was residing prior to his seeking chiropractic care for his LBP.

A physical exam was performed after history taking. The exam did not reveal any spinal deformities, and his gait was normal. There were no overt limitations in active thoracolumbar range of motion although thoracolumbar extension reproduced his characteristic lower back and thigh pain. The thoracolumbar paraspinal regions were mildly tender to palpation with moderately guarded musculature, while spinal percussion did not elicit sharp pain. Lower extremity dermatomal testing revealed asymmetrical sensory findings with diminished sensation over the left lateral leg and foot (i.e., L5-S1 dermatomes), while myotome testing and deep tendon reflexes were normal and symmetrical. Upper motor neuron lesion screening was performed via ankle clonus, Hoffman sign, and Trömner sign, which were all normal. The Slump test was negative for reproduction of lower extremity radicular pain, while the Slump test and the Kemp test did reproduce his characteristic lower back pain.

The combination of clinical features reported by the Veteran supported a preliminary diagnosis of suspected chronic CES likely secondary to underlying degenerative and stenotic changes. Differential diagnoses in-

cluded myelopathy secondary to canal stenosis or lumbar radiculopathy with associated groin hypoesthesia requiring further diagnostic clarification. Although the physical exam did not reveal overtly concerning findings, the progressive and debilitating symptom pattern consistent with CES, combined with the high-risk nature of this condition, warranted immediate referral to the emergency department (ED) for further diagnostic evaluation. Chiropractic treatment was withheld during this initial visit. The Veteran was thoroughly counseled regarding the potential for CES and its serious neurological sequela if not identified and treated in a timely fashion. A same-day lumbar spine MRI was performed at a non-VA ED, which showed severe left and moderate right neuroforaminal stenosis at L5-S1 vertebral segments with mild canal stenosis throughout the lower lumbar spine (Figures 2-3 and Table 1). The ED also placed a neurosurgery consult, which was ultimately declined on the basis that his lumbar spine MRI findings were considered non-surgical. While the neuroforaminal stenosis shown on MRI may have accounted for the patient's L5-S1 thigh pain and reduced leg sensation, the mild central canal stenosis did not provide a clear structural explanation for the bladder dysfunction or groin hypoesthesia; this combination of features is consistent with scan-negative CES.

Table 1.
*Lumbar spine MRI findings on the date of the chiropractic intake**

Impression	
1.)	The conus medullaris and cauda equina are unremarkable.
2.)	Multilevel degenerative changes of the lumbar spine, most pronounced at L5-S1.
3.)	L5-S1: Severe left and moderate right neural foraminal stenosis.
4.)	L4-L5: Mild bilateral neural foraminal stenosis.
5.)	L3-S1: Mild central canal stenosis.

*Full MRI report available in *Appendix 1*

Following the determination by neurosurgery that this Veteran was a non-surgical candidate, he returned to the chiropractic clinic with a strong desire to initiate treatment. At this second visit, he explained how his LBP, groin hypoesthesia, and daily urinary dysfunction had continued, unchanged. These collective clinical features, along with the MRI results, justified an updated diagnosis of scan-negative CES with concurrent radiculopathy, complicated by various psychosocial factors.

The informed consent process heavily emphasized the risks for progressive neurological deficits and included instructions that any worsening of bladder function, groin hypoesthesia, or lower extremity weakness necessitated an immediate return to the ED. Informed consent also emphasized how treatment would be adapted to involve limited force to minimize risk. He verbalized an understanding of these risks and expressed a strong desire to proceed with treatment. A modified approach to treatment was initiated during this second visit, emphasizing a cautious safety-first approach to care. A multimodal treatment approach was used, which included patient education on his condition and features that should prompt emergency follow-up, soft-tissue therapy, application of moist heat, and patient tolerated flexion-distraction applied to the lumbo-pelvic region. More forceful treatments involving the lumbar spine were intentionally deferred to observe the patient's response to less forceful approaches.

One week later, the patient returned for his third visit. He denied any adverse events or worsening neurologic features. He reported similar symptoms as the previous visit but noted a mild-to-moderate reduction of his lower back and leg pain for two to three days after his prior visit, and he attributed this improvement to the flexion-distraction treatment. This multimodal treatment approach was continued at visit three without modification.

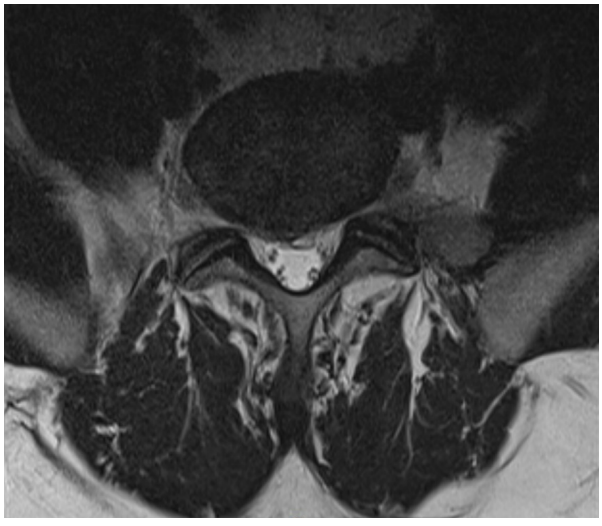
The fourth visit occurred two weeks later, as the patient had missed his appointment that was scheduled for the prior week. He reported a marginal lasting reduction in his LBP to a 7/10 intensity, accompanied by a decreased frequency of radiating leg pain and fewer episodes of bladder dysfunction. Groin hypoesthesia and sexual dysfunction continued, unchanged. A repeat lower extremity neurological exam was unchanged from his initial visit. Considering the patient's symptomatic improvement, high-velocity, low-amplitude (HVLA)

A: T1-weighted lumbar spine MRI **B**: T2-weighted lumbar spine MRI

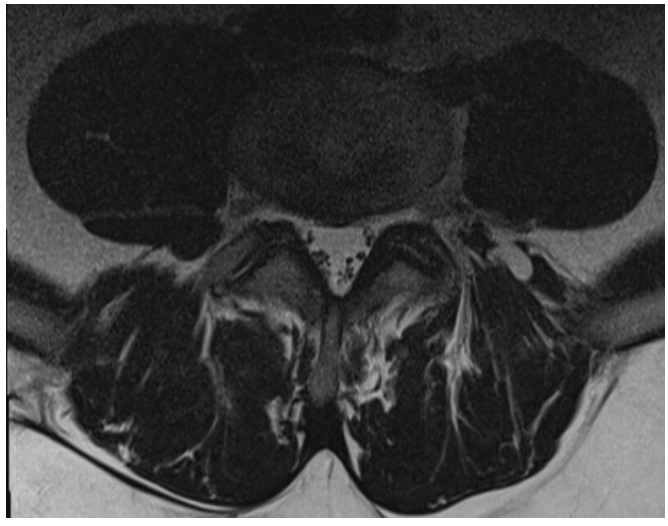
Figure 2.
Sagittal lumbar spine MRI images on the date of chiropractic intake.

lumbar spinal manipulation was judiciously introduced into the established treatment approach with controlled force application, guided by patient tolerance. The rationale for incorporating HVLA was based on the patient's preference, the absence of compressive pathology on MRI (i.e., scan-negative status), and his demonstrated neurological stability following less forceful treatments.

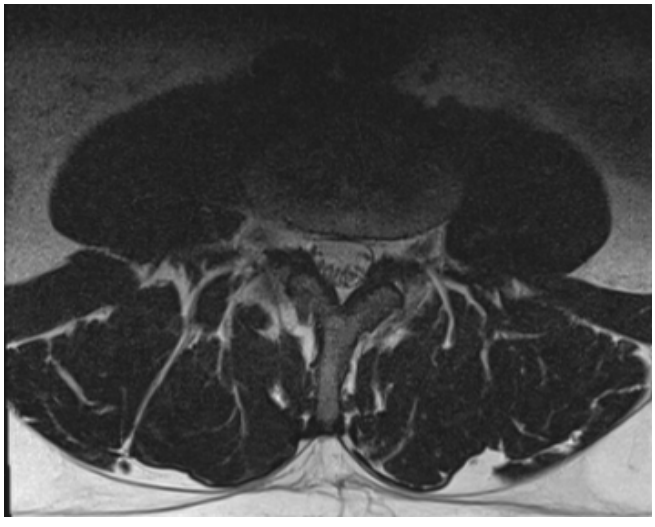
The following week, the patient returned for his fifth visit, which involved a planned re-evaluation. An updated Back Bournemouth questionnaire resulted in a score of 29/70, which represented a 41% clinical improvement from his baseline score of 49/70. Improvements from baseline were also observed in the Defense and Veterans Pain Rating Scale (DVPRS) outcomes and his pain intensity was a 5/10, representing a clinically meaningful reduction in pain compared with his initial pain intensity of 8/10 at the chiropractic intake (Table 2).[13–15] He also reported a complete resolution of his leg pain and bladder dysfunction as well as mild improvement of his groin hypoesthesia. Updated neurological exam findings remained unchanged with continuation of diminished sensation in the left L5 and S1 dermatomes. Multimodal treatment continued at this fifth visit, unchanged from the previous visit.



A: L5 intervertebral disc level



B: L4 intervertebral disc level



C: L3 intervertebral disc level

Figure 3.
Axial T2-weighted lower lumbar MRI images on the date of the chiropractic intake.

At his sixth visit, the patient continued to report an improvement in back and thigh pain and now denied any bladder dysfunction. He also explained how his groin hypoesthesia was improving, stating how he had a "70% return in sensation," with only minor residual burning and tingling. Enhanced sexual function was also described, secondary to this improved sensation. He denied any adverse effects from the prior treatments, and chiropractic care continued, unchanged from the previous visit.

One week later, he returned for his seventh visit. His symptoms were unchanged from the previous visit apart from continued improvement of his groin sensation; he now described a complete return of normal groin sensation. He also noted how he felt these improvements coincided with improvements to his psychological well-being and overall quality of life. Chiropractic treatment was again provided on this seventh visit without modification. Following this seventh visit, the patient was transitioned

Table 2.
Outcome measures at baseline and reevaluation.

Outcome Measure	Baseline	Reevaluation	Improvement
DVPRS			
Pain Intensity	8/10	5/10	38%
Pain Interfering with Activity	8/10	3/10	63%
Pain Interfering with Sleep	7/10	4/10	43%
Pain Affecting Mood	9/10	2/10	78%
Pain Contributing to Stress	9/10	3/10	67%
Back Bournemouth Questionnaire			
Total Score	49/70 (70%)	29/70 (41%)	41%

DVPRS, Defense and Veterans Pain Rating Scale (0/10 = None, 10/10 = As bad or as much as possible)

to an as-needed status (i.e., PRN); however, a telephone follow-up call was scheduled for two weeks to monitor his status.

Following unsuccessful contact at this two-week follow-up, it was discovered that the patient had recently been incarcerated. Communication was established via the county jail's online communication system. While incarcerated, the patient experienced a significant exacerbation of his symptoms, with axial and radicular pain returning to his baseline 8/10 intensity. Additionally, he noted the recurrence of significant groin hypoesthesia and bilateral lower extremity paresthesia but denied any recurrence of bladder dysfunction. He attributed his symptom recurrence to various environmental and psychosocial stressors, specifically limited mobility, increased anxiety, and sleeping on a rigid detention bunk. Postural recommendations and self-management strategies were provided via the facility's communication system. These recommendations included frequent positional changes, supine pelvic tilts, and wall squats. The patient was further instructed in self-directed lumbar decompression and traction maneuvers specifically adapted for his restricted environment (Figures 4 and 5). The patient's release date from the detention facility remains uncertain at the time of writing this report. However, he was advised to return to the chiropractic clinic for reevaluation and possible initiation of a renewed course of care upon his release.

Discussion

There is a lack of robust guidance regarding the optimal management of chronic scan-negative CES, but a safety-first approach is recommended when managing individuals with progressive neurological deficits.[8] Chiropractors are instructed to exercise extreme caution and guardedness when patients exhibit signs or symptoms suggestive of CES, as a delay in neurosurgical referral risks serious neurologic consequesnces.[16] It is vital that clinicians distinguish between scan-positive and scan-negative presentations of CES, as the appropriate management strategies differ significantly between the two subtypes. This report highlights the nuance diagnosis of chronic scan-negative CES, and the potential role chiropractors may play for such cases in an interdisciplinary setting.

Clinicians should screen for red flags by identifying CES-associated signs and symptoms that include severe LBP, radicular pain, altered bowel or bladder function, reduced sensation in the perineal region, loss of sexual function, hyporeflexia, or sensorimotor loss in the lower extremities. Given their limited diagnostic accuracy, identification of such red flag findings should prompt fur-

Figure 4.
Lumbar decompression positioning

Figure 5.
Lumbar self-traction

ther evaluation via an immediate MRI.[17,18] This limitation is underscored by the findings that senior neurosurgical trainees demonstrated only 56% accuracy in predicting scan-positive CES based solely on clinical findings.[19] It is also important to avoid overreliance on imaging, as discrepancies between clinical assessment and radiologic findings may lead to overtreatment or mismanagement. This diagnostic caution is supported by evidence that clinical findings and radiological data fail to correlate in over 50% of suspected CES cases.[20] It has been suggested that patients who display progression of CES symptoms over a few hours or days should undergo immediate imaging (i.e., same day), whereas CES symptoms progressing over a period of weeks should receive urgent imaging (i.e., within five days).[8,21] An informed clinical diagnosis of scan-positive CES requires the integration of clinic-

al signs and symptoms with imaging evidence of cauda equina compression, a finding that warrants emergency surgical decompression to prevent permanent neurological deficits.[22] Although the optimal timing of surgical intervention remains debated, the current standard is decompression within 48 hours.[23,24]

While scan-positive CES is well-characterized with a conceptually straightforward pathology and clear treatment protocol, scan-negative CES represents a complex deviation from this norm. Even though the pathophysiology of scan-negative CES remains poorly understood, a retrospective study suggests that scan-negative patients often exhibit an underlying vulnerability, demonstrated by high rates of psychiatric comorbidities and functional neurological disorders.[11] In vulnerable individuals, acute pain may trigger a predisposition toward functional disorders, resulting in recurrent physical symptoms that mimic an emergent surgical condition. The authors of this report theorize that this patient's high level of psychosocial stressors (e.g., housing instability and mental health conditions) may have served as an underlying amplifier for his chronic CES symptoms. Understanding the nuances of scan-negative CES is crucial for validating patient symptoms, communicating the underlying cause, and effectively addressing the comorbidities that may perpetuate the problem. Approximately 11% of undiagnosed patients enter a cycle of recurrent acute episodes and resource-intensive negative scans,[11] while the underlying functional vulnerability remains unaddressed.

Clinicians should recognize that overlapping conditions may complicate the presentation of clinical features suggestive of CES, while maintaining a safety-first approach to avoid the potentially devastating outcomes of delayed investigation. Differential diagnoses to consider in patients with CES symptoms include inflammatory, vascular, infective, medication-induced, neoplastic and neurodegenerative disorders.[25] Conus medullaris syndrome (CMS) is an uncommon condition that subtly mimics CES, due to overlapping clinical features. CMS results from injury at the conus medullaris, which is at the T12–L2 spinal level, whereas CES arises from insult to the nerve roots below the conus medullaris. The differentiation of these conditions via contrast-enhanced MRI is essential to establish an accurate diagnosis and guide effective management.[26] Similarly, an uncommon inflammatory cause of CES is spinal arachnoiditis, characterized by inflammation of the arachnoid membrane surrounding the cauda equina, in contrast to the more common compressive etiologies such as disc herniation.[27]

In this case, an ED referral was warranted based on the emergence of bladder dysfunction and progression of the patient's chronic symptoms. After ruling out cauda equina compression, a safety-first approach to chiropractic care involved detailed symptom-focused questioning and frequent neurological monitoring to ensure stability of neurologic symptoms. Close observation is recommended to detect any progression of CES-like symptoms for providers involved in treating back or leg-pain; specifically: changes in bowel or bladder function, sexual function, saddle anesthesia, or lower extremity weakness.[21] This case describes the cautious and graded application of manual treatments, informed by the patient's clinical response, and preferences. The authors of this report hypothesize that such manual therapies may have improved pelvic hemodynamics or reduced venous congestion in the cauda equina, which is a proposed mechanism in functional or ischemic forms of CES, but acknowledge that this remains highly speculative.[28]

Patients with LBP commonly seek care from chiropractors, making the failure to diagnose CES a significant source of medicolegal liability for the chiropractic profession.[29] However, the literature indicates that spinal manipulation is not an established risk factor for causing CES and is medically accepted as a conservative treatment option for lumbar disc herniation.[29,30] Although existing research on the use of spinal manipulation in patients with chronic CES is extremely limited, a recent study identified no increase in risk of CES, bladder catheterization and bowel incontinence among adults with lumbar spine disorders receiving spinal manipulative therapy.[31]

In this particular case, meaningful improvements were observed following a carefully monitored course of care. The improvements observed in this individual case cannot be attributed directly to chiropractic treatment and likely reflect natural symptom fluctuations as well as the combined effects of multidisciplinary care, particularly mental health services, contributing to psychosocial stabilization. Nonetheless, this case highlights the importance of vigilant red flag recognition, timely referral, and

interdisciplinary collaboration when managing patients with suspected chronic CES in chiropractic settings.

Limitations

Case reports inherently have several limitations. This report describes a single course of chiropractic care, and the observed outcomes may not be generalizable to other clinical situations. This case involved a multimodal treatment approach over an extended period with various uncontrolled factors, which may have influenced the clinical course. Most importantly, this report is not designed to establish the safety or efficacy of the described clinical approach and should not be considered evidence to support its application in other cases of CES.

Summary

This report describes the chiropractic evaluation and management of a U.S. Veteran with chronic LBP and progressive neurological features consistent with chronic scan-negative CES. Prompt recognition of red flag findings, including bladder dysfunction, groin hypoesthesia, and sexual dysfunction, led to appropriate referral for emergency imaging and facilitated interdisciplinary co-management.

Under a safety-first framework of close neurological monitoring, the patient achieved meaningful improvements in pain, function, and neurological symptoms. These improvements were associated with enhanced physical and psychosocial well-being. Although the management of chronic CES remains poorly defined in the literature, this case suggests that chiropractors may safely contribute to the interdisciplinary management of selected scan-negative CES patients, when red flags are appropriately identified, referral pathways are respected, and treatment is individualized. Further research is needed to clarify best practices for interdisciplinary management of chronic CES and the role of conservative care.

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Authors' contributions

SH delivered clinical care, recorded outcomes and obtained the patient's consent for publication. SH, CBR, and SMS developed the study design and coauthored the manuscript. Each of the authors met criteria for authorship and approved the final submission.

References

1. Miller J, West J, Khawar H, Middleton R. Cauda equina syndrome. Br J Hosp Med. 2005;84(11):1–7. doi:10.12968/hmed.2023.0012
2. Rider LS, Marra EM. Cauda Equina and Conus Medullaris Syndromes. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2026 Jan 7].
3. Lavy C, Marks P, Dangas K, Todd N. Cauda equina syndrome-a practical guide to definition and classification. Int Orthop. 2022;46(2):165–169. doi:10.1007/s00264-021-05273-1
4. Hoeritzauer I, Wood M, Copley PC, Demetriades AK, Woodfield J. What is the incidence of cauda equina syndrome? A systematic review. J Neurosurg Spine. 2020;32(6):832–841. doi:10.3171/2019.12.SPINE19839
5. Gardner A, Gardner E, Morley T. Cauda equina syndrome: a review of the current clinical and medico-legal position. Eur Spine J. 2011;20(5):690–697. doi:10.1007/s00586-010-1668-3
6. Chu ECP, Trager RJ. Prevalence of Serious Pathology Among Adults with Low Back Pain Presenting for Chiropractic Care: A Retrospective Chart Review of Integrated Clinics in Hong Kong. Med Sci Monit Int Med J Exp Clin Res. 2022;28:e938042. doi:10.12659/MSM.938042
7. Gitelman A, Hishmeh S, Morelli BN, Joseph SA, Casden A, Kuflik P, et al. Cauda equina syndrome: a comprehensive review. Am J Orthop Belle Mead NJ. 2008;37(11):556–562.
8. Comer C, Finucane L, Mercer C, Greenhalgh S. SHADES of grey – The challenge of "grumbling" cauda equina symptoms in older adults with lumbar spinal stenosis. Musculoskelet Sci Pract. 2020;45:102049. doi:10.1016/j.msksp.2019.102049
9. Kalichman L, Cole R, Kim DH, Li L, Suri P, Guermazi A, et al. Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine J. 2009;9(7):545–550. doi:10.1016/j.spinee.2009.03.005

10. Olmarker K, Rydevik B, Hansson T, Holm S. Compression-induced changes of the nutritional supply to the porcine cauda equina. J Spinal Disord. 1990;3(1):25–29.
11. Hoeritzauer I, Pronin S, Carson A, Statham P, Demetriades AK, Stone J. The clinical features and outcome of scan-negative and scan-positive cases in suspected cauda equina syndrome: a retrospective study of 276 patients. J Neurol. 2018;265(12):2916–2926. doi:10.1007/s00415-018-9078-2
12. Lim DJ. Atypical intradural extramedullary spinal schwannoma causing cauda equina syndrome: A case report and literature review. Int J Surg Case Rep. 2023;108:108396. doi:10.1016/j.ijscr.2023.108396
13. Polomano RC, Galloway KT, Kent ML, Brandon-Edwards H, Kwon KN, Morales C, et al. Psychometric Testing of the Defense and Veterans Pain Rating Scale (DVPRS): A New Pain Scale for Military Population. Pain Med Malden Mass. 2016;17(8):1505–1519. doi:10.1093/pm/pnw105
14. Boyer CW, Lee IE, Tenan MS. All MCIDs Are Wrong, But Some May be Useful. J Orthop Sports Phys Ther. 2022;52(6):401–407. doi:10.2519/jospt.2022.11193
15. Salaffi F, Stancati A, Silvestri CA, Ciapetti A, Grassi W. Minimal clinically important changes in chronic musculoskeletal pain intensity measured on a numerical rating scale. Eur J Pain. 2004;8(4):283–291. doi:10.1016/j.ejpain.2003.09.004
16. Haldeman S, Rubinstein SM. Cauda equina syndrome in patients undergoing manipulation of the lumbar spine. Spine. 1992;17(12):1469–1473. doi:10.1097/00007632-199212000-00005
17. Dionne N, Adefolarin A, Kunzelman D, Trehan N, Finucane L, Levesque L, et al. What is the diagnostic accuracy of red flags related to cauda equina syndrome (CES), when compared to Magnetic Resonance Imaging (MRI)? A systematic review. Musculoskelet Sci Pract. 2019 ;42:125–133. doi:10.1016/j.msksp.2019.05.004
18. Greenhalgh S, Finucane L, Mercer C, Selfe J. Assessment and management of cauda equina syndrome. Musculoskelet Sci Pract. 2018;37:69–74. doi:10.1016/j.msksp.2018.06.002
19. Bell DA, Collie D, Statham PF. Cauda equina syndrome: what is the correlation between clinical assessment and MRI scanning? Br J Neurosurg. 2007;21(2):201–203. doi:10.1080/02688690701317144
20. Fairbank J, Hashimoto R, Dailey A, Patel AA, Dettori JR. Does patient history and physical examination predict MRI proven cauda equina syndrome? Evid-Based Spine-Care J. 2011;2(4):27–33. doi:10.1055/s-0031-1274754
21. Finucane LM, Downie A, Mercer C, Greenhalgh SM, Boissonnault WG, Pool-Goudzwaard AL, et al. International Framework for Red Flags for Potential Serious Spinal Pathologies. J Orthop Sports Phys Ther. 2020;50(7):350–372. doi:10.2519/jospt.2020.9971
22. Karikaran A, Carroll AH, Benn L, Okorie N, Bellaire CP, Puvanesarajah V, et al. Cauda Equina Syndrome: A Review of Classification, Diagnosis, Treatment, and Best Practices. JBJS Rev. 2025;13(2). doi:10.2106/JBJS.RVW.24.00156
23. Bulloch L, Thompson K, Spector L. Cauda Equina Syndrome. Orthop Clin North Am. 2022;53(2):247–254. doi:10.1016/j.ocl.2021.11.010
24. Kapetanakis S, Chaniotakis C, Kazakos C, Papathanasiou JV. Cauda Equina Syndrome Due to Lumbar Disc Herniation: a Review of Literature. Folia Med (Plovdiv). 2017;59(4):377–386. doi:10.1515/folmed-2017-0038
25. Hoeritzauer I, Stanton B, Carson A, Stone J. "Scan-negative" cauda equina syndrome: what to do when there is no neurosurgical cause. Pract Neurol. 2022;22(1):6–13. doi:10.1136/practneurol-2020-002830
26. Nersesyan N, Brun Vergara ML, Reda A, Fazio Ferraciolli S, Lucato L, Torres C. Pathology of the conus medullaris and cauda equina. Beyond the usual suspects. Insights Imaging. 2025;16(1):225. doi:10.1186/s13244-025-02117-z
27. Maillard J, Batista S, Medeiros F, Farid G, Santa Maria PE, Perret CM, et al. Spinal Adhesive Arachnoiditis: A Literature Review. Cureus. 2023;15(1):e33697. doi:10.7759/cureus.33697
28. Genevay S, Atlas SJ. Lumbar spinal stenosis. Best Pract Res Clin Rheumatol. 2010;24(2):253–265. doi:10.1016/j.berh.2009.11.001
29. Trager RJ, Baumann AN, Perez JA, Dusek JA, Perfecto RPT, Goertz CM. Association between chiropractic spinal manipulation and cauda equina syndrome in adults with low back pain: Retrospective cohort study of US academic health centers. PloS One. 2024;19(3):e0299159. doi:10.1371/journal.pone.0299159
30. Oliphant D. Safety of spinal manipulation in the treatment of lumbar disk herniations: a systematic review and risk assessment. J Manipulative Physiol Ther. 2004;27(3):197–210. doi:10.1016/j.jmpt.2003.12.023.
31. Trager RJ, Baumann AN, Perfecto RPT, Goertz CM. Chiropractic spinal manipulative therapy versus physical therapist-led exercise and the risk of cauda equina syndrome in adults with lumbar disc herniation, stenosis, or radiculopathy. PM R. 2026. doi:10.1002/pmrj.70071.

Appendix 1.
Full radiology report from the lumbar spine MRI on the date of the chiropractic intake.

Reason for Study: Rule out cauda equina in patient with gradually progressive urinary incontinence and erectile dysfunction, intensifying back and thigh pain, normal gait, and a negative upper motor neuron screen
Technique: Multiplanar, multisequence T1-weighted and fluid-sensitive sequences of the lumbar spine from T12 to S2 before and after administration of intravenous contrast.
Comparisons: none
Findings
Spinal Canal: The conus terminates at L1. The conus medullaris and cauda equina are unremarkable. No enhancing masses within the spinal canal. No epidural abscess. Alignment: 3 mm retrolisthesis of L3 on L4. Bone Marrow: Five non-rib-bearing lumbar vertebral bodies are assumed. No gross fractures or bone lesions. No bone marrow replacement or abnormal enhancement. Disk Levels/Facets: T12-L1: Unremarkable. L1-L2: Disk desiccation. Small disk bulge. No significant spinal canal or foraminal narrowing. L2-L3: Disk desiccation. Small disk bulge and facet hypertrophy. Minimal spinal canal stenosis. No significant foraminal stenosis. L3-L4: Disk desiccation and facet hypertrophy with disk bulge, resulting in mild spinal canal stenosis. No significant foraminal stenosis. L4-L5: Disk bulge and facet hypertrophy with minimal spinal canal stenosis. Mild bilateral foraminal stenosis. L5-S1: Disk bulge with superimposed right eccentric disk protrusion. Moderate disk space height loss with disk desiccation. Mild spinal canal stenosis severe left and moderate right foraminal stenosis. Musculature: Normal. No edema, abnormal enhancement, or fatty atrophy.